The immunomodulator IL-10 consistently suppresses NO production in XS-52 cells although to a small extent, as in macrophages. IL-10 inhibits antigen presentation through inhibition of B7(Chang et al. (1995) *Eur J lmmunol* 25:394–398), a costimulatory molecule, expressed with MHC-I. A decrease in NO production secondary to IL-10 may mean that NO partly mediates the effects of IL-10. Also, IL-10 treated cells showed lower levels of iNOS as opposed to cells treated with L-NAME, though both substances inhibit NO production.

Example 7: Nitric Oxide is Toxicfor Melanocytes

Melanocyte susceptibility to NO was examined using NO donor compounds and NO released by a Langerhans cell-like cell line.

Sodium nitroprusside (SNP) is a donor of NO in aqueous solution. Melanocyte lysis was seen in the presence of 0.01–1 mM SNP over 24 hrs, quantified by chromium release. Chromium release was seen to be both time- and SNP dose-dependent, with more chromium being released from melanocytes at a higher SNP-concentration and longer time course. Maximum chromium release of up to 80–90% was seen at 16 hrs after addition of 1 mM SNP.

Langerhans cells (LC) express inducible NOS and produce large amounts of NO. Because LC lie in close proximity to melanocytes in the epidermis, it was hypothesized that the large amounts of NO produced by LC may affect melanocyte function and survival, resulting in pathological manifestations. Co-culture of an LC-like cell line (XS cells) with melanocytes followed by the induction of iNOS by LPS resulted in melanocyte cell death. As melanocytes do not express iNOS, LPS had no effect on melanocytes stimulated with LPS in the absence of XS cells. Melanocyte lysis was also seen when cocultures were performed across Transwells®, with no direct cell-cell contact between XS cells and melanocytes. Thus LC-induced melanocyte death was dependent on a diffusible factor consistent with NO. To confirm that this substance was indeed NO, cocultures were performed in L-arginine deficient media (reversible by addition of L-arginine) or in the presence of NO quencher, reduced hemoglobin. Melanocyte toxicity was remarkable reduced under both conditions. These results indicate an NO-dependent interaction between LC and melanocytes. Thus, nitric oxide from LC and NO donors is cytotoxic for melanocytes.

Equivalents

Those skilled in the art will be able to recognize, or be able to ascertain using no more than routine experimentation, numerous equivalents to the specific procedures described herein. Such equivalents are considered to be within the scope of this invention and are covered by the following claims.

What is claimed is:

1. A method of treating a subject for a condition characterized by excess pigmentation comprising: selecting a subject in need of skin lightening; and admistering to the subject a treatment which increases the level of NO in the skin thereby lighting the skin of the subject.

2. A method of treating a subject for a condition characterized by a lack of pigmentation comprising: selecting a subject in need of skin darkening; and administering to the subject, a treatment which reduces the level of NO in the skin, thereby lightening the skin of the subject.

3. The method of claim **2**, wherein said condition is: vitiligo; post-inflammatory hypopigmentation; or idiopathic guttate hypomelanosis (IGH).

4. The method of claim **2**, wherein the treatment includes the administration of a compound which inhibits the level of NO in the skin of the subject.

5. The method of claim **4**, wherein the treatment includes the administration of an inhibitor of NO synthase or an NO scavenger.

6. The method of claim **1**, wherein the treatment includes the administration of a compound which increases the level of NO in the skin of the subject.

7. The method of claim **6**, wherein the treatment includes the administration of an NO donor compound.

8. The method of claim **1**, wherein the condition is post-inflammatory hyperpigmentation.

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